March 7, 1867.

WILLIAM BOWMAN, Esq., Vice-President, in the Chair.

In accordance with the Statutes, the names of the Candidates for election into the Society were read, as follows:—

Patrick Adie, Esq.
Alexander Armstrong, M.D.
† William Baird, M.D.
John Ball, Esq.
Henry Charlton Bastian, M.D.
Samuel Brown, Esq.
Francis T. Buckland, Esq.
Lieut.-Colonel John Cameron, R.E.
Frederick Le Gros Clark, Esq.
Professor Robert Bellamy Clifton.
Joseph Barnard Davis, M.D.
† W. Boyd Dawkins, Esq.
Henry Dircks, Esq.
Baldwin Francis Duppa, Esq.
William Esson, Esq.
Alexander Fleming, M.D.
Peter Le Neve Foster, Esq.
Sir Charles Fox.
Edward Headlam Greenhow, M.D.
† Albert C. L. G. Günther, M.D.
† Julius Haast, Esq., Ph.D.
† Captain Robert Wolseley Haig, R.A.
† Daniel Hanbury, Esq.
Augustus George Vernon Harcourt, Esq.
Edmund Thomas Higgins, Esq.
Jabez Hogg, Esq.,
† John Whitaker Hulke, Esq.
† Edward Hull, Esq.
James Jago, M.D.
Professor Thomas Hayter Lewis.
† Edward Joseph Lowe, Esq.
David Macloughlin, M.D.
Professor Nevil Story Maskelyne.
John Matthew, Esq.
George Matthey, Esq.
† James Robert Napier, Esq.
Thomas Nunneley, Esq.
Admiral Erasmus Ommaney.
James Bell Pettigrew, M.D.
Charles Bland Radcliffe, M.D.
John Russell Reynolds, M.D.
† Benjamin Ward Richardson, M.D.
† J. S. Burdon Sanderson, M.D.
Edward Henry Sieveking, M.D.
† Henry T. Stainton, Esq.
James Startin, Esq.
John Stewart, Esq.
Major James Francis Tennant, R.E.
Colonel Henry Edward Landor Thuillier, R.A.
† Charles Tomlinson, Esq.
Rev. Henry Baker Tristram.
Cromwell Fleetwood Varley, Esq.
William Sandys Wright Vaux, Esq.
Edward Walker, Esq.
Professor J. Alfred Wanklyn.
Edward John Waring, M.D.
Henry Wilde, Esq.
Samuel Wilks, M.D.
Professor William Parkinson Wilson.
James Young, Esq.
Colonel Henry Yule, R.E.

"On the Influence exerted by the Movements of Respiration on the Circulation of the Blood." Being the CROONIAN LECTURE for 1867, delivered by Dr. J. BURDON SANDERSON.

(Abstract).

The purpose of the lecture was to show that the explanation usually given by physiologists of the mode in which the respiratory movements of the thorax influence the force and frequency of the contractions of the heart can no longer be entertained.

The doctrine usually taught in this and other countries is stated as follows in one of the most recent text-books:—"During the act of expiration the frequency of the pulse is considerably augmented, whilst the line of mean pressure rapidly rises, indicating increased tension in the arterial walls. . . . . During the act of inspiration, on the contrary, the pulsation becomes slower, the curves much bolder, and the line of mean pressure gradually falls; for then the blood readily enters the thorax, and, as a consequence, the great veins, capillaries, and arterial walls become comparatively flaccid" (Carpenter's 'Physiology,' 1864, p. 345). Statements to the same effect are to be found in Budge's 'Lehrbuch der Physiologie,' 1862, p. 350; in Kirke's 'Handbook of Physiology,' 1863, p. 129; in Ludwig's 'Lehrbuch,' 1857, vol. ii. pp. 161, 162.

From numerous experiments, in which the respiratory movements and the variation of pressure in the arteries in the dog were recorded simultaneously by mechanical means, the author had arrived at an opposite conclusion, viz. that in natural breathing each expansion of the chest is followed by increase of arterial tension and shortening of the diastolic interval; in other words, that the immediate effect of inspiration is to increase both the force and frequency of the contractions of the heart.

The *experimental method* was as follows:—For the purpose of recording the movement of air in and out of the chest, the animal is caused to breathe through a T-shaped tube, one arm of which is connected with the trachea, while the other remains open. By the stem it communicates with a disk-shaped bag of thin caoutchouc. The resistance afforded to the ingress and egress of air by the tube, although very inconsiderable, is yet sufficient to produce alternate movements of expansion and collapse of the bag. The variations of arterial pressure are measured by a mercurial manometer, differing from that of Poiseuille, in that the attached arm, which is the longer of the two, is of much smaller diameter than the other, the area of the latter being twelve times as great as that of the former. For the purpose of recording the movements of the dynamometer and of the caoutchouc bag, two light wooden levers of the third kind, each 25 inches in length, are used. These work on steel axes, the bearings of which are so contrived that the axis of the arterial lever is directly above that of the respiratory lever, and that both oscillate in the same vertical plane: by vertical rods they are connected, the upper or arterial lever with a cork float

which rests on the surface of the mercury in the wide arm of the dynamometer, the lower with the upper surface of the caoutchouc bag. At their extremities they carry fine sable brushes, by which their movements are inscribed on a roll of paper, to which a horizontal movement is communicated by clockwork. By a mechanical arrangement, which need not be here described, synchronical points can from time to time be marked in the two tracings inscribed simultaneously on the paper by the momentary withdrawal of both brushes. The experiments were of the following nature, dogs being employed throughout.

1. *Experiments as to normal respiration.*—In these experiments (eleven in number) the dynamometer was connected with the femoral artery, while the breathing-tube was connected with the respiratory cavity, either by the trachea, or by means of a mask fixed over the snout. The principal results were as follows:—

Experiment 1.—Respirations per minute, 9; pulsations, 108. Mean arterial pressure 6·2 inches. The tracings show that each respiratory act is divisible into two parts; two-fifths being occupied by thoracic movements, the remainder by the pause. Of the former, two-thirds correspond to inspiration, one-third to expiration. During the pause the arterial pressure gradually sinks, the commencement of inspiration being immediately followed by an increase of pressure, which becomes still more marked during expiration, but again subsides at its completion. The interval between each two succeeding contractions of the heart is seen to be three times as great in those pulsations which immediately *follow* expiration as in those which precede it.

The other experiments of the series were of a similar nature. In some the relative length of the respiratory intervals and the regularity of the pulsations rendered it more easy to judge of the precise relation between the two tracings than in others, but in all the results were in complete accordance with those above stated. Even when the frequency of breathing was such that three pulsations corresponded to one respiration (experiment 4), it was observed that the diastolic interval which immediately followed expiration was twice as long as either of the other two. In one case the respiratory tracing showed that the mode of breathing was peculiar: inspiration was separated from expiration by a pause of considerable duration, during which the arterial pressure declined and the pulse was retarded.

2. *Experiments for the purpose of determining whether the resistance afforded by the* T*-tube to the passage of air in and out of the chest exercise any modifying influence on the results.*—It was obvious that this end could be best attained by observing the effect of increasing the resistance; for by so doing, any modifying influence exercised by it would become more obvious. With this view a series of observations were made on the same animal (under the influence of morphia), in which the resistance was gradually increased by inserting plugs of various sizes into the aperture of the T-tube. The tracings showed that even when the aperture was so diminished

as to produce marked dyspnœa, and great exaggeration of the movements of respiration, it was observed as distinctly as before that the increase of force and frequency of the pulse were increased by the prolonged inspiratory efforts of the animal.

3. *Experiments showing that when the respiratory cavity is completely closed (as by plugging the trachea), the relation between the respiratory movements of the chest and the arterial pressure is reversed.*—The process of death by apnœa may be divided into two stages; the first extending from the moment of occlusion to the cessation of the struggles of the animal and the supervention of apparent insensibility, the second terminating with the extinction of the circulation. In order to observe the characters of the respiratory movements and those of the heart during these two stages, it was necessary to substitute a mercurial manometer for the caoutchouc bag. It was then seen that at first the respiratory movements increase in amplitude without altering in character; but towards the end of the first minute, when the animal begins to struggle, they become irregular, and each struggle is accompanied by strong *expulsive* efforts, during which the mercury in the dynamometer oscillates violently and rises to an enormous height. At the commencement of the second stage, when the animal becomes tranquil, the respiratory movements assume a different character, become almost exclusively inspiratory (gasping), and much more regular. They occur, however, at longer and longer intervals, until they finally cease. As regards the relation between the oscillations of the two manometers, the tracings show distinctly that throughout the whole process they are strictly coincident, both as to the *time* of their occurrence and their extent. Hence it may be concluded that the extraordinary elevation of arterial pressure which has been long known to occur during the second minute in death by apnœa, is not due, as was supposed by Dr. Alison and Dr. John Reid, to obstruction of the capillary vessels, either pulmonary or systemic, but to the violence of the respiratory efforts. The cavity of the chest being closed, the force exercised by the respiratory muscles expresses itself in variations of tension of the enclosed air, which are communicated through the intra-thoracic arteries to those outside of the chest, producing those violent oscillations of the dynamometer which have been referred to.

In support of this inference, it was shown that in an animal under the influence of woorara (when all respiratory movement ceases, while those of the heart are unaffected), the process of apnœa is not only of greater duration, but is not attended with any of those peculiar disturbances of the circulation which have been hitherto attributed to capillary obstruction. The gradual extinction of the force of the contraction of the heart is indicated by a slow and uninterrupted subsidence of the arterial pressure.

4. *Experiments for the purpose of ascertaining in how far the influence exercised by the respiratory movements on the heart in ordinary breathing are chemical.*—For this purpose observations were made on animals which

had been allowed to respire a limited quantity of air (50–100 cubic inches) for a sufficiently long time to ensure the complete cessation of all appreciable reaction of its oxygen on the circulating blood. In this form of apnœa insensibility is not produced until from ten to fifteen minutes after the experiment. As in ordinary suffocation, it is associated with a marked change in the mode of breathing. All expiratory efforts cease, and the animal respires by gasps, each of which is separated from its successor by a pause of variable duration. Under these circumstances, when unquestionably all chemical reaction is out of the question, the effect observed is of the same nature as in ordinary breathing, the only difference being that, in consequence of the length of the intervals and the absence of expiratory effort, it is much more obvious. The moment after inspiration commences, the mercurial column is jerked up by a succession of forcible contractions of the heart.

5. *Experiments showing that in artificial respiration, when the mechanism is reversed, the chemical conditions remaining the same, the mechanical effect is correspondingly modified; and that if the blood is venous, a chemical effect is produced by each injection of air into the lungs, which, although of the same nature, requires a much longer time for its production.*—If, in an animal under the influence of woorara, artificial respiration be discontinued until the arterial pressure sinks several inches, and then air is injected, even in small quantity, no immediate effect is observed excepting a momentary increase of arterial pressure coincident in time with the expansion of the lungs; but after the lapse of six or seven seconds, the heart begins to beat with extreme frequency, rapidly raising the mercurial column until a pressure is attained equal or superior to that originally existing. The length of the time which intervenes between this event and its antecedent is in itself sufficient to show that the relation between the two cannot be mechanical. This is proved by the observation that, if hydrogen be substituted for air in the experiments, no effect is produced.

6. *Experiments showing the relation between the thoracic movements and the arterial pressure after section of the pneumogastric nerves.*—Section of the pneumogastric nerves in the neck, besides its well-known effect in retarding the breathing and accelerating the contractions of the heart, alters the mode of the respiratory movements. The chest is unnaturally dilated even during the pause. Inspiration is performed slowly and with effort, and terminates in a sudden expiratory collapse. The heart not only contracts more frequently, but more forcibly, the arterial pressure rising several inches of mercury. Under these conditions it is observed (1) that the arterial pressure tends to increase during the slow inspiration, and to decline during the pause, and (2) that a more rapid increase of tension occurs simultaneously with expiration. But (3) no variation is observed of the frequency of the pulsations; and (4) all the effects are much less marked than in the normal animal. These peculiarities are to be attributed to the extreme rapidity of the heart's action, to the permanent

distension of the thoracic veins, and to the violence of the expiratory movements.

Theoretical exposition of the mechanical influence of the respiratory movements on the circulation.—(1) It has been demonstrated by Donders that the elastic contents of the chest have at all times a tendency to shrink to a smaller bulk than that of the cavity in which they are contained, so that the viscera within the thorax are constantly distended in a degree which varies according to its ever-varying capacity. As, however, they are not equally elastic, they yield to this distension unequally. When the chest enlarges, the lungs yield most, the veins and heart, in a state of relaxation, next; the contracting heart and the arteries scarcely expand at all. (2) If the veins contained air and communicated with the atmosphere, they would fill as rapidly as the lungs; actually their expansion is much slower. Hence the first effect of inspiration is to increase the proportion of thoracic space occupied by the lungs, by which they become relatively more distended than the other organs. So soon, however, as the veins and auricles have time to fill, equilibrium is more or less restored. (3) Hence it follows (*a*) that the dilatation of the chest in inspiration aids the expansion of the heart during diastole and of the thoracic veins; and (*b*) that these events cannot occur simultaneously with their cause, but must follow at an interval varying according to the condition of the circulation. (4) Other things being equal, the force and frequency of the contractions of the heart are increased by whatever causes accelerate its diastolic impletion. The more rapidly the cavities fill the shorter must be its period of relaxation, the more vigorous its systole, and consequently the greater the arterial pressure. (5) The effect of thoracic expansion on the intra-thoracic veins varies both as regards its degree and the time of its occurrence. Both kinds of variation depend on the velocity of the circulation and the pressure existing in the veins outside of the chest. When the systemic veins are distended, the circulation rapid, and the arterial resistance in consequence diminished, the heart almost empties itself at each contraction, and the expansion of the chest fills the thoracic veins and the relaxed heart with great rapidity. In the opposite case, when the systemic veins are comparatively empty, the cavities of the heart fill slowly, and discharge themselves imperfectly on account of the excessive arterial resistance. (6) Hence *the effect of inspiration* in facilitating the diastolic impletion of the auricles, and consequently *in increasing the frequency and force of the heart's action, varies directly as the velocity of the circulation, inversely as the arterial pressure.*

Conclusions.—1. In natural breathing the influence exercised by the thoracic movements on the heart is entirely mechanical. So long as the respiration is tranquil, variations of air-pressure in the bronchial tubes and vesicles of the lungs do not materially affect the arterial pressure; but violent expiratory movements are accompanied by simultaneous increase of vascular tension.

2. When the respiratory orifices are closed, the variations of blood-pressure in the arteries are synchronical with those of air-pressure in the respiratory cavity, and take place in the same direction.

3. The increased action of the heart which results from chemical changes produced in the circulating fluid by exposure to air, is of the same nature as the mechanical effect of inspiration, both being indicated by increased arterial tension and acceleration of the pulse. The former may be distinguished from the latter (*a*) by the length of time required for the production of the effect, and (*b*) by its dependence on a previous venous condition of the blood.

4. Hence the influence of the thoracic movements on those of the heart may be either directly mechanical, as in suffocation, indirectly mechanical, as in ordinary breathing, or chemical.

March 14, 1867.

Lieut.-General SABINE, President, in the Chair.

The following communications were read:—

I. "Note on Mr. Merrifield's New Method of calculating the Statical Stability of a Ship." By W. J. Macquorn Rankine, C.E., LL.D., F.R.S. Received February 22, 1867.

On the 24th of January, 1867, a paper was read to the Royal Society by Mr. C. W. Merrifield, F.R.S., Principal of the Royal School of Naval Architecture, showing how, by determining the radii of curvature of the locus of the centre of buoyancy or "metacentric involute" of a ship in an upright position and at one given angle of inclination, a formula may be obtained for calculating to a close approximation her moment of stability at any given angle of inclination, on the assumption that the metacentric involute can be sufficiently represented by a conic.

It has occurred to me that the latter part of the calculation in Mr. Merrifield's method might be simplified by assuming for the approximate form of the metacentric involute, not a conic, but the *involute of the involute of a circle*; the locus of its centres of curvature, or "metacentric evolute," being assumed to be the involute of a circle.

The involute of the involute of a circle is distinguished by the following property. Let r be the radius of the circle, ρ_0 that radius of curvature of the involute of the involute which touches the involute at its cusp, and ρ another radius of curvature of the same curve making the angle θ with the radius ρ_0; then

$$\rho = \rho_0 + \frac{r\theta^2}{2}. \quad \ldots\ldots\ldots \quad (1)$$

Having found, then, the radii of curvature of the metacentric involute in